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The Metabolic Effects of Scorpion Sting in Children

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SUMMARY

The metabolic disturbance is one of the major problems that may aggravate the cardiovascular, neurological, and gastrointestinal manifestation of a scorpion sting. This work aims to evaluate of clinical manifestations of these cases. For this purpose, 200 cases of scorpion sting patients admitted to the medical center of Sabha received from different hospitals in the South of Libya were examined clinically as well as tested for metabolic changes. Patient's ages ranged from 3 to 15 years of both sexes. The tested parameters included blood glucose level, serum potassium, sodium and bicarbonates, and blood pH. Blood pressure, ECG, and relevant clinical manifestations were also examined. Fifty normal children with matched ages and sexes served as the control group and were examined for the same parameters. Serum potassium and glucose increased significantly in the patient group. Serum bicarbonate and blood pH also were changed in patients. The serum sodium was slightly less in the patients but was not significant.

INTRODUCTION

Scorpion stings are common in North Africa. The great majority of cases in otherwise healthy subjects can be conservatively treated by adequate analgesia, local application of ice, and a period of observation dictated by the clinical findings. Particular care should be taken in the assessment of the very young and old and those with pre-existing morbidities, such as hypertension and diabetes. In children, a scorpion sting is a life-threatening emergency. Most of the children with severe envenomation die because of the toxin, whereas it is a relatively less serious condition in adults [1].

Scorpion envenomation results in a severe autonomic storm with massive release of catecholamines, increased angiotensin, and inhibition of insulin secretion. These hormonal alterations could be responsible for the pathogenesis of a variety of clinical manifestations. Under these conditions, scorpion envenomation results in a syndrome of fuel energy deficit and an inability to utilize the existing metabolic substrate by vital organs causing multi-organ system failure and death [2].

MATERIAL & METHODS

Two hundred patients of both sexes (53 % males and 47% females) with an age ranging from 3 to 15 years (mean 9.3 ± 4.48 years) admitted to the

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Medical Center of Sabha in the period between 2017 to 2022 were chosen for the study. Fifty healthy children's age (mean age 8.84 ± 5.2 years) and sex (52 % males and 48% females) are used as control.

All patients had a definite history of sting (by their relatives and/ or local manifestations). All these stung children and control had no history of allergy, previous sting, pulmonary or cardiac disease, or recent infection. All patients and control were subjected to full clinical examination including general, cardiovascular, chest, and CNS examination.

Blood samples were withdrawn using a scalp vein set (Shanchuan) and blood was collected in sterile test tubes. Blood glucose was measured using (Quick touch apparatus).

Serum sodium, potassium, bicarbonates, and blood pH were measured using (Rapid Lab 855, Bayer) blood gas analyzer.

The data was tabulated and analyzed using a statistical package for Social Science (SPSS) version 11.0 for personal computers (PC).

RESULTS

Serum sodium was significantly reduced in the patient group than in the control group ($p< 0.05$) where it was about 131.5 in the patient while it was 135 in the control group, table (1). Serum potassium was significantly lower in the patient group (3.3 ±0.44 MEq/L) than in the control group (3.7 ± 0.4 MEq/L) ($p< 0.05$) Table 1. Serum bicarbonate was insignificantly less (18.4 ± 10.4 MEq/L) in the patient group when compared with the control group (20.5 ± 1.2 MEq/L) ($p > 0.05$) Table (1). The blood pH did not show any significant change in both groups where it was 7.37 in patients and 7.33 in the control group. Blood glucose increased with a highly significant difference in the patients (140 ± 58.2 mg/dl) when compared with control subjects (112.58 ± 22.60 mg/dl) ($p < 0.01$) table (1).

Table 1: Analysis of Variance (ANOVA) of the serum parameters in the patients and control groups.

Value / Parameter	Mean		±SD		F	P
	C	P	C	P		
s. Sodium	135	131.5	3.5	4.9	5.43	0.019
s. Potassium	3.7	3.3	0.4	0.44	6.267	0.015
s. Bicarbonates	20.5	18.4	1.2	10.4	1.179	0.282
pH	7.33	7.37	0.17	0.051	0.326	0.570
Glucose	112.58	140	22.60	58.2	7.619	0.008

P<0.05 the difference is statistically significant

P>0.05 the difference is statistically insignificant

SD Standard deviation

C Control

P Patients

There was a significant correlation between the delay of patients' arrival at the hospital (the time lapse between the sting and measurement of the parameters) and the blood pH, systolic and diastolic blood pressure (table 2).

Table 2: Two-tailed Pearson Correlation parameters of patients group between the correlations.

	delay	nat	kt	Pico3	pH	Glu
Delay Pearson Correlation	1	0.017	0.045	0.138	0.371	0.029
Sig. (2-Tailed)		0.919	0.793	0.424	+ve 0.028*	0.863
Nat Pearson Correlation	0.017	1	0.029	0.120	0.001	0.137
Sig. (2-Tailed)	0.919		0.820	0.357	0.995	0.288
Kt Pearson Correlation	0.045	0.029	1	0.057	0.169	0.132
Sig. (2-Tailed)	0.793	0.820		0.665	0.196	0.307
Pico3 Pearson Correlation	0.138	0.120	0.057	1	0.077	0.099
Sig. (2-Tailed)	0.424	0.357	0.665		0.556	0.448
pH Pearson Correlation	0.371	0.001	0.169	0.077	1	0.953
Sig. (2-Tailed)	0.028	0.995	0.196	0.556		0.011
Glu Pearson Correlation	0.029	0.137	0.132	0.099	0.049	1
Sig. (2-Tailed)	0.863	0.288	0.307	0.448	0.710	
Sbp Pearson Correlation	0.126	0.119	0.023	0.206	0.175	0.953
Sig. (2-Tailed)	0.240	0.504	0.896	0.241	0.331	-ve 0.011
Dbp Pearson Correlation	0.189	0.009	0.266	0.114	0.375	0.237
Sig. (2-Tailed)	0.076	0.958	0.128	0.520	-ve 0.031	.0177

N.B. dbp diastolic blood pressure

Sbp systolic blood pressure

P < 0.05 is significant

P > 0.05 is insignificant

As regards the clinical data, the blood pressure was elevated in 33% of cases (67 cases) (for the age). In comparison, it was lower than normal the age in 25 % of cases (50 cases) while the remaining cases were normotensive. Vomiting was mild to moderate 3 – 4 times) in 56 cases (28%) of cases and severe (repeated) in 13% (26 cases). Sweating was mild to moderate in 60 cases (30 % of cases) and severe in 12 cases (6 % of cases) (table 3).

Table 3: Incidence of clinical manifestations of scorpion sting.

Signs	Grade	N. of cases	Percent (%)
Vomiting	Absent	118	59
	Mild & moderate	56	28
	Severe	26	13
	Total	200	100
Sweating	Absent	132	66
	Mild to moderate	56	28
	Severe	12	6
	Total	200	200

Scorpion sting causes a wide range of effects, ranging from local pain and paresthesia at the site of the sting to cardiotoxicity, pulmonary edema, encephalopathy, and severe metabolic derangement [3]. Scorpion venom constitutes low molecular weight polypeptides. The long-chain polypeptides cause stabilization of the voltage-dependent sodium channels in the open position, leading to continuous, prolonged, and repetitive firing from the somatic, sympathetic, and parasympathetic neurons. This repetitive firing results in autonomic and neuromuscular over-excitation symptoms and prevents normal nerve impulse transmission. Meanwhile, the short polypeptide neurotoxin blocks the potassium channel [4].

Because of its action on neurons, scorpion venom causes a massive release of neurotransmitters such as epinephrine, norepinephrine, acetylcholine, glutamate, and aspartate, which are responsible for most of the systemic signs and symptoms of scorpion envenomation [2]. The sympathetically induced secretion of rennin due to the action of venom ultimately results in the activation of angiotensin II which causes the release of aldosterone from the adrenal gland [5].

Scorpion venom stimulates the secretion of glucagon and cortisol suppresses insulin secretion [6], and reduces the level of circulating thyroxine (T4) and tri-iodotyrosine (T3) [7].

The significant decrease in serum sodium level was due to excessive vomiting and sweating [8]. However, sodium loss was counteracted by high renal conservation of sodium [9] and the release of aldosterone because of scorpion venom [5]. These opposing mechanisms resulted in a modest but significant decrease in serum sodium from 135 MEq/L for the control to 132.9 MEq/L for the patients. This result is in agreement with that of [10] who reported hyponatremia in nearly all of the scorpion stings reviewed over 2 years.

The significant decrease in serum potassium level was due to several factors, large quantities of potassium can be lost in vomiting [11], together with potassium loss under the effect of the released aldosterone [8] and adrenocortical hormones [11]. In cases of stress as in trauma, self-limited hypokalemia occurs in 50 – 60 % of cases related to enhanced release of epinephrine [8]. This result is in contrast to that of [12] who reported an increase in the serum potassium in case of scorpion sting and attributed this increase to prolonged hyperglycemia and acidosis. However, acidosis was not a prominent feature in our work.

The serum bicarbonate level and blood pH did not differ significantly in patients and controls ($p>0.05$). This result may be due to several opposing mechanisms. Loss of hydrogen ions following hydrochloric acid loss due to repeated vomiting will be compensated by a rise in plasma bicarbonate level [13]. The rise in blood pH will stimulate respiratory compensation [11]. The compensatory mechanism and the associated potassium depletion and excess aldosterone release will contribute to lowering the blood pH and bicarbonate level [14].

Scorpion venom itself may cause metabolic and respiratory

acidosis probably because of hypoxia [15]. These mechanisms which operate in an opposing manner might have resulted in nonsignificant changes in serum bicarbonate level and blood pH.

The serum glucose levels were significantly higher in patients than in control. Hyperglycemia resulting from scorpion envenomation was noticed earlier by many investigators, where liver glycogenolysis stimulated by excessive release of catecholamines seemed to be the major factor contributing to the rise in blood glucose. In addition, scorpion venom itself was found to cause suppression of insulin secretion [7]. The excessive release of glucocorticoids and glucagons increases the hepatic output of glucose and its anti-insulin activity on the peripheral tissues. Finally, excessive catecholamines decrease insulin release which may be an additional factor in causing hyperglycemia [16].

As regards the correlation between parameter 2- tailed Pearson correlation revealed a positive correlation between blood pH and delay, and a negative correlation between delay and systolic and diastolic blood pressure. The positive correlation with delay shows that the more delay the more is the normalization of the blood pH. This may be because long delay permits the operation of more compensatory mechanisms which require longer time to operate as renal compensation and other buffering systems and hormonal factors [17].

The negative correlation between delay and blood pressure indicates that the longer the delay the worse the blood pressure. This effect may be due to failure of the initial compensatory mechanisms, loss of extracellular fluid by the excessive perspiration and vomiting, and accumulation of large amounts of potent vasodilator substances most probably prostaglandins particularly in children [18,19].

In conclusion, the metabolic effects of scorpion stings are serious complications and some of them may be aggravated by the delay in interference. Further studies are required to determine the implications of these metabolic disturbances on the clinical course and severity of the condition and their prognostic value in predicting the outcome of the cases.

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